UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/563,571	06/08/2006	Naveen Chandra Bisht	47677-0001-00-US (220114)	1031

55694 7590 03/18/2009

DRINKER BIDDLE & REATH (DC)
1500 K STREET, N.W.
SUITE 1100
WASHINGTON, DC 20005-1209

EXAMINER	
FOX, DAVID T	
ART UNIT	PAPER NUMBER
1638	
MAIL DATE	DELIVERY MODE
03/18/2009	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/563,571	BISHT ET AL.	
	Examiner	Art Unit	
	David T. Fox	1638	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *8/21/08 & 12/11/08*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-30* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-30* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *06 January 2006* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *8/25/08*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

Restriction/Election

The Restriction Requirement of 27 June 2008 has been WITHDRAWN, upon further consideration. Claims 1-30 are pending and examined in the instant Office action.

Amendment Compliance with 37 CFR 1.121(c)

Applicant traverses the Notice of Non-Compliant Amendment of 17 November 2008, asserting that there is no requirement that font changes must be indicated in an amendment.

The Examiner notes that not all of the text had font changes. The terms that the Examiner highlighted were either scientific names of plants or bacteria, e.g., *Brassica, Brassica juncea, Agrobacterium*; or gene names, e.g., *hpt, bar, barnase, barstar*. It is conventional in the art to italicize such names. Thus, the choice of font for these names is not trivial.

The original claims correctly recited all of the scientific names or gene names in italics, while the amendment of 21 August 2008 de-italicized some but not all of these names. In the interest of compact prosecution, the Examiner is examining all of the claims. Applicant is encouraged to submit claims where all of the scientific names and gene names are italicized, in keeping with art-recognized usage.

Indefiniteness

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 4, 7-10, and 12-30 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. Dependent claims are included in all rejections.

Claim 4 is indefinite in its recitation of "gene is…barstar and protease inhibitors" which is confusing in its equation of genes with proteins. The following amendment would obviate this rejection:

In claim 4, line 1, replace "is" with ---encodes a protein---.

Claims 7-8 and 24-25 are indefinite in their recitation of "gene…characterized by the…sequence" which is confusing. It is unclear whether the sequence is representative of the class of genes, or whether the gene actually comprises the exact sequence. It is unclear whether the sequences are exemplary or required. The following amendments would obviate this rejection:

In claims 7-8, line 2, replace "characterized by" with ---having--- or ---comprising---.

In claims 24-25, line 2, replace "is characterized by" with ---has--- or ---comprises---.

Claims 9-10 and 26-27 are confusing in their equation of genes with promoters. The following amendments would obviate this rejection:

In claim 9, insert ---promoters--- before the period.

In claim 10, replace "TA29" with ---a TA29 promoter---, and replace "A9" with ---an A9 promoter---.

In claim 26, replace "TA29" with ---a TA29 promoter---.

In claim 27, replace "A9" with ---an A9 promoter---.

Claim 12 is indefinite in its recitation of "MMV" and "FMV" as it is unclear to what these abbreviations refer. If basis in the specification exists, Applicant should recite the complete name of these terms, followed by the abbreviations in parentheses. Moreover, the recitation of "promoter...is...MMV and "FMV" is indefinite for its equation of viruses with promoters.

Claim 13 is indefinite in its recitation of "plant and parts or seeds thereof which contain...the construct", which employs improper Markush terminology per MPEP 2173.05(h). Furthermore, it is unclear whether all of the recited species contain the construct. The following amendments would obviate this rejection:

In line 1, replace "and" with ---or---, and insert --- , each of--- after "thereof".

Claim 16 is indefinite in its recitation in part (a) of parentheses, as it is unclear whether the parenthetical subject matter is required or exemplary. The claim fails to positively recite required claim elements. The following claim amendments would obviate this rejection:

In claim 16, part (a), lines 2-3, replace the parentheses with commas.

In line 2 of part (a), insert ---promoter--- after "which".

In line 3 of part (a), replace "and" with ---said restorer gene sequence---.

Claim 17 is indefinite in its recitation of "preferably" which fails to positively recite a required claim element. It is unclear whether the subsequently recited plant species is required or merely exemplary.

The proposed amendments above have been presented in the format of Examiner's amendments for brevity. All claim amendments performed by Applicant should comply with 37 CFR 1.121(c).

Obviousness

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1-2, 5-6 and 11-15 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Flasinksi (US 2006/0191038 effectively filed 18 July 2002)** in view of **Fabijanski et al (US 6,162,964),** further in view of **Shah et al (PNAS 79: 1022-1026 issued 1982)**.

The claims are drawn to a DNA construct comprising a first and second transgene each encoding the same restorer protein, wherein the transgenes have different nucleotide sequences to avoid gene silencing and are under the control of two different male reproductive tissue-specific promoters, wherein one of the promoters may be the same as the promoter driving a male sterility gene, wherein the DNA constructs also comprise a 3' polyadenylation signal, wherein the DNA construct may also comprise a selectable marker gene including a *bar* or *hpt* gene under the control of a CaMV 35S promoter; and transformed dicotyledonous or monocotyledonous plants comprising them, including the dicot *Brassica juncea*.

Flasinksi teaches the advantages of a DNA construct comprising a first and second transgene each encoding the same protein, wherein the transgenes have different nucleotide sequences to avoid gene silencing, wherein the transgenes may be under the control of male tissue-specific promoters including pollen-specific promoters, wherein different promoters may be used for each transgene, wherein the construct also comprises a 3' polyadenylation signal, wherein herbicide resistance genes including the *bar* gene may be employed, and wherein monocotyledonous plants such as corn or wheat, or dicotyledonous plants such as cotton or *Arabidopsis* or *Brassica* or tobacco, may be transformed. Flasinski teaches the monocot and dicot species-specific requirements for codon usage, wherein such preferred codon usage may be employed to create synthetic genes encoding the same protein.

Flasinksi also suggests the use of the construct to encode any protein conferring any agronomically important trait, including pollination control.

See, e.g., page 1, paragraphs [0005], [0007-0008]; page 2, paragraph [0015]; page 3, paragraphs [0073] and [0077-0078]; page 4, paragraphs [0085-0092]; page 5, paragraph [0122]; pages 9-10, paragraph [0178]; pages 11-15, paragraphs [0187-0198] and Tables 1-4; page 19, paragraph [0211]; page 20, paragraph [0218]; page 21, paragraph [0224]; pages 22-23, paragraphs [0233-0246]; page 25, paragraph [0255]; pages 28-30, paragraphs [0272-0283].

Flasinksi does not explicitly teach a DNA construct comprising two transgenes each encoding the same restorer protein.

Fabijanski et al teach tobacco and *Brassica* plant transformation with a male sterility-inducing gene and a male fertility-restoration gene, each under the control of male tissue-specific promoters, including the L4 or L10 or L19 *Brassica* promoters which do not share high sequence homology. Fabijanski et al suggest that the male sterility-inducing gene encode an antisense RNA corresponding to a housekeeping gene encoding a protein required by all plant cells, said antisense RNA-encoding gene being under the control of a male tissue-specific promoter. Fabijanski et al teach the use of various marker genes including herbicide resistance genes or antibiotic resistance genes, under the control of constitutive promoters like the CaMV 35S promoter.

Fabijanski et al specifically suggest that the male sterility gene encode antisense RNA to an actin gene, and that the restorer gene encode the actin protein. Fabijanski et al suggest the use of different promoters for multiple

transgenes, and also suggest the use of the same promoter for the male sterility gene and the male fertility gene. Fabijanski et al teach the *Agrobacterium*-mediated transformation of the dicots tobacco and *Brassica napus*, and suggest the transformation of a variety of agronomic crop species including monocots or the dicot *Brassica juncea*.

See, e.g., Figures 3a, 3b and 3d; column 9, lines 14-47 and 66-67; column 10, lines 1-24 and 57-67; column 11, lines 1-23; column 12, lines 19-28; column 13, lines 7-35; column 14, lines 4-29; column 15, lines 55-67; column 16, lines 10-48; column 17, lines 4-17 and 38-44; column 22, lines 8-25 and 32-39; column 24, lines 8-24 and 35-52; column 28, lines 34-62; column 29, lines 14-23; column 30, lines 36-67; column 31, line 61 through column 32, line 11; column 36, line 60 through column 37, line 44; column 38, lines 1-8 and 32-36; column 42, line 64 through column 43, line 5; column 55, lines 5-40; column 59, line 24 through column 60, line 25.

Shah et al teach an actin gene sequence (see, e.g., page 1023, Figure 2).

It would have been obvious to one of ordinary skill in the art to utilize the DNA construct comprising two agronomic trait-conferring genes encoding the same protein but comprising different gene sequences, under the control of two different tissue-specific promoters as taught by Flasinksi; and to modify that construct by incorporating the fertility restorer genes and male tissue-specific promoters taught by Fabijanski et al, wherein the male sterility gene encodes antisense RNA to the actin gene taught by Shah et al, and wherein the male fertility restorer gene encodes the actin protein via the sense gene taught by

Shah et al; given the suggestion to do so by Flasinksi and Fabijanksi et al. It would have been further obvious to employ as one of the male fertility restorer promoters the same promoter as that used by the male sterility gene, as suggested by Fabijanski et al. Choice of transformable *Brassica* species would have been the optimization of process parameters.

Claims 1-2, 5-6 and 9-15 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Flasinksi** (US 2006/0191038 effectively filed 18 July 2002) in view of **Fabijanski et al** (US 6,162,964), further in view of **Shah et al** (PNAS 79: 1022-1026 issued 1982), further in view of **Jagannath et al (2001, Molecular Breeding 8: 11-12, Applicant submitted)**.

The claims are drawn to the DNA construct above wherein one male tissue-specific promoter is the TA29 promoter and the other promoter is the A9 promoter, and plants transformed therewith including *Brassica juncea* plants.

Flasinski in view of Fabijanski et al, further in view of Shah et al, teach a DNA construct comprising two fertility restorer genes encoding the same actin protein but differing in nucleotide sequence, each under the control of two different male tissue-specific promoters as discussed above, but do not teach the TA29 or A9 promoters.

Jagannath et al teach *Brassica juncea* transformation with male sterility genes under the control of either the TA29 or A9 promoters (see, e.g., Abstract).

It would have been obvious to one of ordinary skill in the art to utilize the DNA construct comprising two fertility restorer genes encoding the same actin protein but differing in nucleotide sequence, each under the control of two

different male tissue-specific promoters, as taught by Flasinski in view of Fabijanski et al, further in view of Shah et al; and to modify that construct by incorporating the TA29 and A9 male tissue-specific promoters taught by Jagannath et al. The substitution of equivalent elements which perform the same function would have been obvious to one of ordinary skill in the art, in the absence of evidence to the contrary.

Claims 1-6 and 11-15 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Flasinksi** (US 2006/0191038 effectively filed 18 July 2002) in view of **Fabijanski et al** (US 6,162,964), further in view of **Jofuku et al (1989, The Plant Cell 1: 1079-1093)**, further in view of **Stevenson et al (1986, Nucleic Acids Research 14: 8307-8330)**.

The claims are drawn to a DNA construct comprising two fertility restorer genes under the control of two different male tissue-specific promoters, wherein the two restorer genes encode the same protein but differ in nucleotide sequence, wherein one of the promoters is the same one as that used in a male sterility gene, wherein the male sterility gene encodes a protein and the male fertility gene encodes a protease inhibitor.

The teachings of Flasinski have been summarized above.

Flasinski does not teach the particularly claimed male sterility or male fertility genes.

The teachings of Fabijanski et al have been summarized above. Fabijanski et al also suggest a male sterility gene encoding a protease such as

trypsin, and a male fertility restorer gene encoding trypsin inhibitor. See, e.g., column 31, lines 34-42 and 46-58; column 58, lines 15-50.

Jofuku et al teach the sequence of a trypsin inhibitor gene (see, e.g., page 1081, Figure 2).

Stevenson et al teach the sequence of a trypsin gene (see, e.g., page 8312, Figure 2).

It would have been obvious to one of ordinary skill in the art to utilize the DNA construct comprising two agronomic trait-conferring genes encoding the same protein but of different gene sequence, under the control of two different tissue-specific promoters as taught by Flasinksi; and to modify that construct by incorporating the fertility restorer genes and male tissue-specific promoters taught by Fabijanski et al, wherein the male fertility restorer gene comprises the trypsin inhibitor gene taught by Jofuku et al, and wherein the male sterility gene comprises the trypsin gene taught by Stevenson et al; given the suggestion to do so by Flasinksi and Fabijanksi et al. It would have been further obvious to employ as one of the male fertility restorer promoters the same promoter as that used by the male sterility gene, as suggested by Fabijanski et al. Choice of transformable *Brassica* species, or choice of known source of trypsin and trypsin inhibitor genes, would have been the optimization of process parameters.

Claims 1-6 and 9-15 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Flasinksi** (US 2006/0191038 effectively filed 18 July 2002) in view of **Fabijanski et al** (US 6,162,964), further in view of **Jofuku et al** (1989, The Plant Cell 1: 1079-1093), further in view of **Stevenson et al** (1986, Nucleic

Acids Research 14: 8307-8330), further in view of **Jagannath et al** (2001, Applicant submitted).

The claims are drawn to the DNA construct above wherein one male tissue-specific promoter is the TA29 promoter and the other promoter is the A9 promoter, and plants transformed therewith including *Brassica juncea* plants.

Flasinski in view of Fabijanski et al, further in view of Jofuku et al and Stevenson et al, teach a DNA construct comprising two fertility restorer genes encoding the same trypsin inhibitor protein but differing in nucleotide sequence, each under the control of two different male tissue-specific promoters as discussed above, but do not teach the TA29 or A9 promoters.

Jagannath et al teach *Brassica juncea* transformation with male sterility genes under the control of either the TA29 or A9 promoters (see, e.g., Abstract).

It would have been obvious to one of ordinary skill in the art to utilize the DNA construct comprising two fertility restorer genes encoding the same trypsin inhibitor protein but differing in nucleotide sequence, each under the control of two different male tissue-specific promoters, as taught by Flasinski in view of Fabijanski et al, further in view of Jofuku et al and Stevenson et al; and to modify that construct by incorporating the TA29 and A9 male tissue-specific promoters taught by Jagannath et al. The substitution of equivalent elements which perform the same function would have been obvious to one of ordinary skill in the art, in the absence of evidence to the contrary.

Claims 1-7, 9, 11-24, 26 and 28-30 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Flasinksi** (US 2006/0191038 effectively filed 18 July

2002) in view of **Fabijanski et al** (US 6,162,964), further in view of **Williams et al (US 5,750,867)** and **Williams et al (1998, SEQ ID NO: 17 of US 5,750,867)**, further in view of **Michiels et al (US 6,372,960, Applicant submitted)**.

The claims are drawn to a DNA construct comprising a first and second transgene each encoding the same restorer protein, wherein the transgenes have different nucleotide sequences to avoid gene silencing and are under the control of two different male reproductive tissue-specific promoters including the TA29 promoter, wherein one of the promoters may be the same as the promoter driving a male sterility gene, wherein the male sterility gene may encode barnase and the male fertility restorer gene may encode barstar, wherein the barstar-encoding gene may comprise SEQ ID NO:1, wherein the DNA constructs also comprise a 3' polyadenylation signal, wherein the DNA construct may also comprise a selectable marker gene including a *bar* or *hpt* gene under the control of a CaMV 35S promoter.

The claims are also drawn to transformed dicotyledonous or monocotyledonous plants comprising the fertility restorer genes, including the *Agrobacterium*-transformed dicot *Brassica juncea*. The claims are also drawn to methods of crossing the restorer plant with a male sterile plant to produce F1 progeny comprising the restorer gene, wherein the presence of the fertility restorer gene is confirmed by molecular analysis and/or pollen viability assays.

The teachings of Flasinksi have been discussed above. Flasinski does not teach male fertility restorer genes including the *barstar* gene.

The teachings of Fabijanski et al have been discussed above. Fabijanski et al also teach the crossing of male sterile plants with male fertile restorer plants to maintain the male sterility gene (see, e.g., column 28, lines 34-62; column 29, lines 14-23). Fabijanski et al also suggest the use of a ribonuclease gene as the male sterility gene, and a ribonunclease inhibitor gene as the male fertility restorer gene (see, e.g., column 31, lines 34-42 and 46-59).

Williams et al (US 5,750,867) teach the crossing of male sterile corn plants, comprising the TA29 or promoter operably linked to a *barnase* gene encoding a ribonuclease, with male fertile restorer corn plants comprising the TA29 promoter operably linked to a *barstar* gene encoding a ribonuclease inhibitor, wherein additional male tissue-specific promoters including the CA55 promoter may be employed, and wherein molecular analysis is performed to confirm the presence of the restorer gene in progeny plants. Williams et al teach that the promoter for the sterility gene and the restorer gene may be the same. Williams et al also teach a selectable marker gene including the *bar* gene or the *hpt* gene (encoding neomycin phosphotransferase) under the control of the CaMV 35S promoter. Williams et al also suggest the application of their method to a variety of transformable agronomic crops including *Brassica napus*.

See, e.g., claims 1 and 5-9; column 2, lines 34-63; column 3, lines 22-49 and 63-67; column 4, lines 1-10; column 5, lines 49-65; column 6, lines 17-24; column 9, line 56 through column 10, line 23; column 14, lines 37-67; column 16, line 15 through column 17, line 23; column 18, lines 20-34; column 20, line 23 through column 22, line 27.

The *barstar* sequence utilized by Williams et al (US 5,750,867) is 99.4% identical to SEQ ID NO:1, with only one mismatch, presumably due to a sequencing error or other obvious variation, as evidenced by Williams et al (1998).

Michiels et al teach a synthetic *barstar* gene encoding the wild-type barstar protein, wherein said synthetic *barstar* gene was modified for codon optimization and enhanced protein expression in plants, wherein the *barstar* gene may be expressed under the control of a variety of male tissue-specific promoters including the TA29 and CA55 promoters (see, e.g., column 2, lines 3-29; column 3, lines 10-31; column 6, lines 46-62; column 7, lines 35-53; column 8, lines 17-24 and 57-67; column 9, lines 1-18; claims 1-11).

It would have been obvious to one of ordinary skill in the art to utilize the DNA construct comprising two agronomic trait-conferring genes encoding the same protein but comprising different gene sequences, under the control of two different tissue-specific promoters as taught by Flasinksi; and to modify that construct by incorporating the fertility restorer genes and male tissue-specific promoters taught by Fabijanski et al, as suggested by each reference.

It would have been further obvious to further modify that construct by incorporating the wild-type *barstar* gene and *barnase* genes, and the TA29 and CA55 male tissue-specific promoters, taught by Williams et al, as suggested by each reference. One of ordinary skill in the art would have recognized the benefits of also utilizing the synthetic *barstar* sequence for enhanced gene expression taught by Michiels et al. One of ordinary skill in the art would have

recognized the benefits of utilizing either molecular analysis taught by Williams et al, and/or pollen viability assays, for evaluating the presence of the fertility restoration gene. Choice of transformable agronomic species, including the corn transformed by Williams et al or the *Brassica* transformed by Fabijanski, would have been the optimization of process parameters.

Claims 1-7, 9-24, and 26-30 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Flasinksi** (US 2006/0191038 effectively filed 18 July 2002) in view of **Fabijanski et al** (US 6,162,964), further in view of **Williams et al (US 5,750,867)** and **Williams et al (1998)**, further in view of **Michiels et al** (US 6,372,960, Applicant submitted), further in view of **Jagannath et al** (2001, Applicant submitted).

The claims are drawn to the DNA constructs and methods above, wherein both the TA29 and A9 promoters are utilized.

Flasinski in view of Fabijanski et al, further in view of Williams et al (US 5,750,867) and Williams et al (1998), further in view of MIchiels et al, teach the claimed DNA constructs and methods as discussed above, but do not explicitly teach the combination of the TA29 and A9 promoters.

Michiels et al also suggests the use of the A9 promoter as one of the male tissue-specific promoters (see, e.g., column 6, lines 46-62).

Jagannath et al teach *Brassica juncea* transformation with male sterility genes encoding barnase and under the control of the TA29 or A9 promoters, as discussed above.

It would have been obvious to one of ordinary skill in the art to utilize the DNA constructs comprising the TA29 promoter operably linked to a wild-type *barstar* gene including SEQ ID NO:1, and further comprising a different male tissue-specific promoter such as the CA55 promoter operably linked to a codon-optimized synthetic *barstar*-encoding sequence, for the restoration of male fertility in a variety of agronomic crop species including *Brassica napus*; as taught by Flasinski in view of Fabijanski et al, further in view of Williams et al (US 5,750,867) and Williams et al (1998), further in view of MIchiels et al; and to modify those DNA constructs by incorporating the TA29 and A9 promoters taught by Jagannath et al, as suggested by Michiels et al, in the absence of evidence to the contrary.

Conclusion

Claims 8 and 25 are deemed free of the prior art, given the failure of the prior art to teach or reasonably suggest plant transformation with the particular *barstar* gene sequence comprising SEQ ID NO:3.

No claim is allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to David T. Fox whose telephone number is (571) 272-0795. The examiner can normally be reached on Monday through Friday from 10:30AM to 7:00PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Anne Marie Grunberg, can be reached on 571-272-0975.

The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/David T Fox/

Primary Examiner, Art Unit 1638

March 13, 2009